Editorials

Hyperventilation and anxiety state[1]

Hyperventilation is common enough, but its range and variety of physiological effects are rarely appreciated. Medical awareness indeed generally stops short at the 'hysterical' fit, with extreme hyperpnoea, tetany and sometimes coma which occasionally enlivens casualty and intensive care units. Tetany, however is one of the least common manifestations; few physicians recognize the more subtle, but infinitely more common syndromes of chronic habitual hyperventilation. These may present with symptoms relating to virtually any organ or system, suggesting, for example, thyroid, cardiac, gastrointestinal, respiratory or central nervous system disease, leading to much fruitless investigation and the accumulation of a dossier more remarkable for size than for clinical relevance. Lewis (1957), in an analysis of 150 cases, found that their original diagnoses numbered over thirty! Hyperventilation can fairly claim to have replaced syphilis as the great mimic. The archetypal syndrome is Da Costa's, currently enjoying a revival as 'mitral valve prolapse syndrome' – the latest in a long line of attempts to attribute a common physiological dysfunction to an anatomical variant which is rarely significant.

White & Hahn (1929) and Kerr *et al.* (1937) first identified these multiple syndromes with hyperventilation, and their observations have since been corroborated by numerous observers whose statistics show it to account for 6–10% of all referrals to specialist clinics (Rice 1950, Engel *et al.* 1947, Lewis 1964, McKell & Sullivan 1947, Tucker 1963, Yu *et al.* 1959). Nevertheless, this common disorder is still not mentioned in standard texts: a surprising omission, given the well known fact that at least one-third of patients seeking advice have no identifiable pathology.

Such patients, generally regarded as neurotic, may present with symptoms suggesting dysfunction or disease of any organ or part of the body. 'Cardiac' manifestations, e.g. palpitations and precordial pains, are particularly common; as are neurological disturbances: dizziness, faintness or blackouts, a great variety of visual disturbance and paraesthesiae affecting many parts of the body, but particularly hands, feet and face. Respiratory complaints, surprisingly, are less common, shortness of breath tending to be overshadowed by general fatigue; but irritable unproductive cough is frequent, as are gastrointestinal symptoms – dysphagia, globus, burping, oesophageal reflux and heartburn. Muscular cramps, 'fibrositis' of neck, shoulders and back are often troublesome but tetany is very rare. Most disturbing are psychic disturbances ranging from tension, through free-floating anxiety to 'unreal' feelings, depersonalization and even hallucinations. The latter two symptoms are seldom volunteered, for there is frequently an unspoken fear of madness. General exhaustion, lack of concentration and diminished performance are almost universal, while sleep disturbance, nightmares, and emotional sweating (armpits and palms) are often found.

Gottlieb (1969), reviewing the incidence of such patients in his general medical oupatient clinic, found that they constituted 40.2% of all referrals and no organic cause could be identified; but he did not consider the possibility of hyperventilation.

Several factors foster the neglect of hyperventilation as a positive diagnosis, e.g. the absence of conspicuous overbreathing. Shortness of breath is seldom the primary complaint. But, most important, is the too ready acceptance of the blanket diagnosis 'neurosis' or 'anxiety state' to cover the inability of physicians to explain multiple symptoms without overt pathology.

Modern neurophysiology, and latterly the use of intracellular microelectrodes, can now supply answers to most of the problems which have previously hindered appreciation of the true role of hyperventilation in anxiety states. It is therefore apposite to review the physiological consequences of hyperventilation; to examine its relation to somatic manifestations of anxiety and to attempt to answer the important question 'Does anxiety cause the symptoms, or do the symptoms cause the anxiety?'

Hyperventilation acts by blowing off excessive quantities of carbon dioxide thus producing respiratory alkalosis. It might seem logical, therefore, to expect that in chronic hyperventilators, one should find an abnormally low level of carbon dioxide in the arterial blood (hypocarbia). Values below the accepted norm are indeed found in about two-thirds of cases (Lum 1976), but in more than one-third – a substantial minority – the level is either equivocal or in the low-normal range. Such inconstancy of relationship between symptoms and objective evidence of chronic alkalosis has led influential

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observers (e.g. Wood 1941) to discount the role of hyperventilation. Wood's view seems to have prevailed in British medicine to the extent that few except the rare and florid forms of hyperventilation are diagnosed.

The diagnosis, however, can be readily proven by a simple provocation test: forced voluntary overbreathing will almost always reproduce symptoms which the patient recognizes within 2–3 minutes. This manoeuvre, besides its diagnostic value, underlines the fact that it is the change in $Paco_2$ rather than the prevailing level which is important. The symptomatic response is evoked as readily when the baseline $Paco_2$ is normal as when it is low.

A low value of carbon dioxide *per se* does not necessarily cause symptoms. The body can learn to adapt asymptomatically to very low levels of $Paco_2$, e.g. in high altitude adaptation. At 14000 feet the mean $Paco_2$ in normals is 27 mmHg, as against 40 mmHg at sea level. This, however, is a long-term adaptation; full acclimatization takes days or weeks. Such adaptive mechanisms cannot protect against short-term fluctuations of $Paco_2$ and it is precisely such transient variations which are the hallmarks of the chronic hyperventilation syndrome.

Rapid, irregular, sighing respirations, with widely varying tidal volumes and, in particular, excessive thoracic movements, have been noted by most observers. The spirometer tracing has been used (Christie 1935) as a diagnostic tool in providing a graphic depiction of the often extravagantly erratic breathing pattern. Christie found inspection of the tracing alone to be diagnostic in 86% of cases of 'neurosis'. Such breathing is inherently unstable; minor stress evokes an exaggerated respiratory response with disproportionate increase in sighs and thoracic movement. Medical consultation is a potent trigger for such tell-tale signs – obvious to the discerning eye and ear, but rarely recorded in the case notes.

Over the last thirty years neurophysiological research has demonstrated the all-pervasive influence of changes in carbon dioxide level on the function of the nervous system. The infrared analyser now provides a ready means of monitoring breath-by-breath variations in arterial Pco_2. Expired air is sampled at the mouth. The air delivered at the end of expiration (i.e. end-tidal air) represents a sample of mixed alveolar air. This, being in equilibrium with pulmonary capillary blood, accurately reflects the arterial Pco_2.

In the normal person, during quiet breathing, continous monitoring of end-tidal Pco_2 shows only minor fluctuations from the mean value. A single, deep breath, however, may lower this by 10 mmHg or more and this may take 20–30 seconds to recover. Voluntary overbreathing for three minutes produces a sharp fall to a low level which is maintained during the overbreathing period, but recovers to normal within a few minutes.

The behaviour of hyperventilators is markedly different. At rest, considerable fluctuations in end-tidal Pco_2 occur, mirroring the fluctuations in tidal volumes; a marked fall occurs with each sigh and recovery takes longer than in the normal, while many show a progressive fall during the observation period. Voluntary overbreathing produces a sharp fall, maintained during the overbreathing period, but on cessation of the manoeuvre the Pco_2 tends to remain low for many minutes; some indeed continue to hyperventilate. Once started they find it difficult to stop.

These are the physiological counterparts of clinical findings: voluntary overbreathing rapidly induces hypocarbic symptoms in normals, but recovery is equally rapid. In hyperventilators familiar symptoms are produced (and recognized) but recovery is delayed and may require recourse to a rebreathing bag, or even the inhalation of 5% carbon dioxide in oxygen.

The symptom complexes attributed variously to anxiety state or to hyperventilation (according to the predilection of the clinician) embrace a wide variety of somatic symptoms, organ dysfunction – particularly cardiac and gastrointestinal – anxiety and disturbance of mental function. The common pathway leading to this plethora of symptoms is the nervous system. Thus, in considering the physiological background it is pertinent to review the known data on the effects of hypocarbia on that system. The subject is extremely complex (*see* Wyke 1963) and only a few relevant and established facts can be mentioned here.

At neuronal level, carbon dioxide acts directly on the nerve cell. Its molecular size allows it to diffuse in and out of the cell even faster than water: it is one of the fastest diffusing molecules. A fall in arterial and cerebrospinal Pco_2 is immediately followed by migration of co_2 from the neurone. The intracellular pH rises, and with it there is a demonstrable increase in neuronal activity and electrical dischage in associated nerve fibres.

Contrary to general belief, the (H+) ion cannot itself diffuse in and out of the neurone, since it is linked to an H_2O molecule, and the diameter of this hydrated proton is greater (9 Ångström units) than that of neuronal pores (6 Ångström units).

This stimulatory effect occurs with quite minor degrees of hypocarbia, e.g. a fall from the norm of 40 mmHg to 35 mmHg. More profound falls produce a change in neuronal metabolism. Then anaerobic glycolysis begins to produce lactic acid

which lowers pH again, with consequent diminution of neuronal activity so that in extreme degrees of hypocarbia the neurone may eventually become inert. Thus the response is biphasic: initially excitation; later, with more profound hypocarbia, progressive depression. Clinically this manifests itself as excitability in the early and mild stages; later, if hyperventilation continues and increases, progressing to exhaustion. In extreme cases, stupor or coma supervene.

There is a specific effect on facilitatory synapses involved in somatic motor reflex arcs, e.g. pupil and tendon reflexes: synaptic transmission is accelerated and reflex time shortened.

In the autonomic system there is a selective depression of parasympathetic activity. (The sympathetic is also depressed, but to a lesser degree.) Hence the subject tends to present a picture of sympathetic dominance: dilated pupils, cold extremities, palmar and axillary sweating, and tachycardia. With more profound hypocarbia these disappear, in similar fashion to the extinction of the stimulation response when mild hypocarbia proceeds to severe.

Thus moderate degrees of hyperventilation – classically in the 'fight or flight' situation – cause increased motor excitability and also increased sensitivity to sensory stimuli: lights appear brighter, sounds louder; photophobia and hyperacusis are not uncommon. The survival value of this complex response is obvious: heightened sensory perception, the muscular system tense and alert, the reflexes quickened. In severe grades of hypocarbia many of these responses become extinguished – hence the phenomenon of paralysis by fright.

Furthermore, carbon dioxide controls the calibre of cerebral arteries. Hypocarbia causes vasoconstriction, and hence cerebral hypoxia. This hypoxia is augmented by a shift to the left of the haemoglobin dissociation curve for oxgyen (Bohr effect), which diminishes both the amount and the rate of transfer of oxygen to tissues. Many of the clinical effects stem from cerebral hypoxia (Lennox *et al.* 1938).

It should be emphasized that most of these neurological effects are produced by falls in $Paco_2$ similar in magnitude to those recorded in the fluctuations typical of hyperventilation. The central nervous system of sufferers is thus repeatedly subjected to hypocarbic stress – a stress felt in all parts of the central and autonomic nervous systems.

The foregoing provides only a brief account of some of the extremely diverse effects of hypocarbia. There is great individual variation in response, particularly in the cerebral vascular reaction. Dizziness and faintness are almost universal in the young; less so in the elderly. Some tolerate with very few symptoms levels of Pco_2 which would produce syncope in others. Dizziness, faintness, visual disturbance and impaired mental performance are closely related to cerebral hypoxia. The compounding of sensory and motor disturbance with hypoxic effects, aptly termed 'a welter of unpleasant bodily sensations', may cause curious mental states: depersonalization is frequent; occasionally hallucinations are experienced, but these are seldom divulged spontaneously lest they should confirm the patient's frequent fear of madness.

A few other clinical effects of hypocarbia should be mentioned. Firstly, it markedly potentiates the effects of alcohol. Hyperventilation is very common in aircraft. The passenger who gets out of hand on a couple of whiskies is a problem familiar to air hostesses. Hyperventilating motorists should therefore be doubly careful not to drink and drive.

Although most forms of perception are heightened, there is one important exception – pain. The complex sensation we recognize as pain is dulled or abolished. This is exemplified in tribal initiation or voodoo rites during ritual mutilation ceremonies. The excitement, singing, dancing and beating of drums induce a crescendo of hyperventilation, during which initiates are enabled stoically to endure otherwise extremely painful procedures. Finally, hyperventilation powerfully aids the induction of hypnosis. Most standard procedures for hypnotic induction employ deliberately induced hyperventilation. In the light of the known neurological effects of hypocarbia, many of the bizarre manifestations of hypnosis can be understood.

It remains to consider why certain individuals become prone to hyperventilate (a perfectly normal reaction) to a degree which causes recurrent or persistent symptoms. Neurological considerations can now leave little doubt that the habitually unstable breathing is the prime cause of symptoms. Why they breathe in this way must be a matter of speculation, but manifestly the salient characteristics are pure habit – excessive sighing, sniffing, nervous cough; and such habits are often family characteristics. Exaggerated thoracic breathing is encouraged by cultural influences such as physical education and sexual display (the chesty he-man and the bosomy blonde). Singers, actors and athletes are trained to make maximal inspiratory efforts with the thorax as well as the diaphragm. Hyperventilation is an occupational hazard with opera singers.

The vast majority of the author's cases have tended to be perfectionist or mildly obsessional. The type A personality (Friedman & Rosenman 1959) is particularly prone in men. These all tend

to drive themselves too hard in setting themselves goals at the limit of their reach.

It is often assumed that anxiety is the chief or only cause of hyperventilation. On the contrary, any change of mood – happiness, laughter, relief, animated conversation, and even watching television – can frequently be the cause. The first attacks commonly follow a purely physical illness. General anaesthesia and operations are potent triggers. The driving personality, addicted to his work, often develops the first attack at weekends or on holiday. Anxiety then develops out of the persistent symptoms. With repetition the response takes on the character of a conditioned reflex (Cannon 1928).

Although Kerr *et al.* (1937) had pointed out that the clinical manifestations of anxiety state were produced by hyperventilation, it was Rice (1950) who turned this concept upside down by stating that the anxiety was produced by the symptoms and, furthermore, that patients could be cured by eliminating faulty breathing habits. Lewis (1964) identified the role of anxiety as a trigger, rather than the prime cause. Given habitual hyperventilation, a variety of triggers, psychic or somatic, can initiate the vicious cycle of increased breathing, symptoms, anxiety arising from symptoms exacerbating hyperventilation and thus generating more symptoms and more anxiety. He claimed a 70% cure rate by breathing re-education.

The present writer's experience – to December 1979, 1735 patients confirmed by respiratory physiological analysis – amply supports this view. More than 1000 patients have received a course of breathing retraining and relaxation in the physiotherapy department. Symptoms are usually abolished within one to six months. Some young patients require only a few weeks while older or more severe cases may take many months: 75% are completely free of all symptoms at 12 months; 20% are left with occasional mild symptoms only, and these do not trouble them. Some of these become asymptomatic later. They lose their anxiety. Only about one in twenty is quite intractable.

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Protecting and improving the health of the public

How can health care be provided at reasonable cost? How can resources be allocated appropriately between cure and care, between services based in institutions and services in the community, and between treatment and prevention? What organizations are required to ensure availability of treatment and help to those who are ill or disabled, and to protect the public from a multitude of threats to health arising from genetic, biological, physical, behavioural and social influences? These questions perplex policymakers all over the world. The Canadian Government was one of the first among developed countries to announce its intention 'to give to human biology, the environment and lifestyle as much attention as it has to the financing of the health care organization so that all four avenues to improved health are pursued with equal vigour' (Lalonde 1974). Recently the Australian Commonwealth Department of Health received a report urging more attention to preventive medicine (Davidson *et al.* 1979). In the same year the US Department of Health, Education and Welfare published a report of the Surgeon General on 'Health Promotion and Disease Prevention' (Surgeon General 1979). The US Report reviews the evidence for the control of risk factors and for

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